



NOTES

BRAIN ISCHEMIA

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Impaired brain function due to lack of blood

TYPES

Focal ischemia

- Occlusion of blood vessel → ↓ perfusion → affected regions damaged
- ↓ oxygen → ischemic stroke
- Blood vessel rupture → hemorrhagic stroke; bleeding inside parenchyma/between brain membranes

Global ischemia

- Cardiac arrest → whole brain hypoperfusion → brain damage

CAUSES

- Atherosclerotic plaque/thrombosis/emboli
- Hypertension
- Blood vessel malformation
- Cardiac arrest, tachycardia, congenital heart problems
- Tumors

SIGNS & SYMPTOMS

- Altered consciousness; weakness; problems with vision, hearing, swallowing; dizziness, vertigo
- See mnemonic for common symptoms

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Visualize trauma, bleeding, skull fracture

MRI

- Visualize hypointense, hyperintense blood clot

MR/CT angiography

- Visualize occlusions, aneurysms

TREATMENT

MEDICATIONS

- Antiplatelet medications (e.g. aspirin/ clopidogrel)
- IV tissue plasminogen activator (tPA)
- Mannitol, other osmotic diuretics
 - ↑ intracranial pressure treatment

SURGERY

- Evacuation of blood clot
- ↑ intracranial pressure treatment

OTHER INTERVENTIONS

- Manage conditions that worsen prognosis (e.g. hyperglycemia, fever)



MNEMONIC: FAST

Common stroke symptoms

Facial drooping

Arm weakness

Speech difficulties

Time: reminder to call emergency services

VISIT...

LANZAROTE
Caliente.COM

EPIDURAL HEMATOMA

osms.it/epidural_hematoma

PATHOLOGY & CAUSES

- Nervous tissue compression due to accumulation of blood in epidural space
- Head trauma → skull fracture → damage of blood vessels through dura mater → extradural blood accumulation → rapid, limited expansion of hematoma due to tight dura adherence at cranial sutures → brain tissue compression → neurological decline

TYPES

Intracranial

- Frontal injuries
 - Anterior ethmoidal artery
- Temporoparietal (most common)
 - Middle meningeal artery
- Occipital
 - Transverse, sigmoid sinus
- Vertex
 - Superior sagittal sinus

Spinal

- Venous plexus of lumbar, thoracic regions

CAUSES

- Neurosurgical procedures complication
- Trauma

Intracranial epidural hematoma

- Head trauma → pterion skull fracture (most common)
- Blood vessel malformations

Spinal epidural hematoma

- Trauma (e.g. lumbar puncture/epidural anesthesia)

RISK FACTORS

- More common in individuals who are biologically male, between 2–60 years
- Pregnancy

- Spontaneous spinal hematoma (very rare)
- Systemic lupus erythematosus
 - Vasculitis, associated with immune system reaction
- Coagulopathies, bleeding diathesis, sickle cell anemia

COMPLICATIONS

- ↑ intracranial pressure
- Supratentorial herniation → compression of arteries → ischemic stroke
- Infratentorial herniation → brainstem compression → heart, respiratory arrest
- Paralysis/sensory loss
- Seizures

SIGNS & SYMPTOMS

- Initial loss of consciousness, lucid state if blood slowly accumulating; delayed neurological deterioration consequence of enlarging hematoma compression
- Intracranial epidural hematoma
 - Broken skull with hematoma
 - Otorrhea/rhinorrhea
 - Altered consciousness
- ↑ intracranial pressure
 - Headache
 - Nausea with vomiting
 - Cushing reflex (↑ blood pressure, ↓ heart rate, irregular breathing)
 - Focal signs (weakness of extremities on opposite side; dilated pupil on injured side due to compression of CN III)
- Spinal epidural hematoma
 - Radicular back pain (resembles pain from herniated discus)
 - Sensory defects
 - Urinary, fecal incontinence

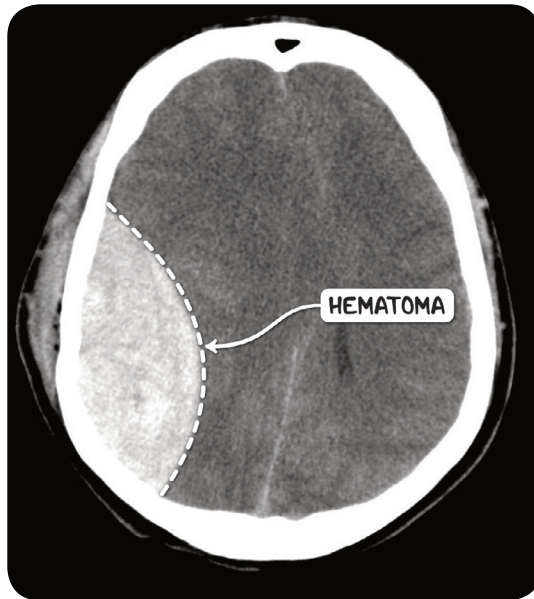


Figure 64.1 A CT scan of the head in the axial plane demonstrating a large epidural hematoma with a classical biconvex shape.

DIAGNOSIS

DIAGNOSTIC IMAGING

X-ray

- Skull fracture

CT scan

- **Hematoma:** typically presents as a **biconvex**, relatively heterogeneous, **high density** mass in the space between skull, brain; **does not cross sutures**
- **Swirl sign:** bleeding into blood clot, diverse hypoattenuated foci
- Assess hematoma volume
- Skull fracture

MRI

- T2-WI: acutely
 - Hypointense blood clot due to deoxyhemoglobin
- T1, T2-WI: in following weeks
 - Deoxy → methemoglobin; hyperintense blood clot
- T1-WI: months later
 - Methemoglobin → hemosiderin; hypointense mass

TREATMENT

MEDICATIONS

- Mannitol, other osmotic diuretics
 - ↑ urine excretion, ↓ intracranial pressure
- Anticoagulation reversal
 - Individuals undergoing surgery, on anticoagulation therapy

SURGERY

- Craniotomy
 - Evacuation of blood mass
- Embolization/ligation of damaged blood vessel
- Trephination (burr-hole)
 - In acute EDH, if neurosurgical procedure delayed
- Laminectomy
 - ↓ blood in spinal epidural hematoma

OTHER INTERVENTIONS

- Observation, nonoperative management
 - Awake, conscious individuals
 - If hematoma volume < 30cm³, thickness < 15mm, midline shift < 5mm

INTRACEREBRAL HEMORRHAGE

osms.it/intracerebral-hemorrhage

PATHOLOGY & CAUSES

- Condition characterized by blood vessels rupture → **intraparenchymal blood accumulation**
- Blood vessel trauma, rupture → creates pool of blood → tissue, surrounding blood vessel compression → hypoxia in downstream tissue → damage due to compression, oxygen lack

CAUSES

Hypertension

- Most common
- Leads to
 - **Atherosclerosis** in large arteries
 - **Hyaline arteriolosclerosis** → focal arterioles necrosis → small wall ruptures → subclinical microbleeds
 - Charcot–Bouchard **aneurysms** (microaneurysms)
- Basal ganglia; thalamus; midbrain; pons; cerebellum primarily affected

Vascular abnormalities

- Cerebral amyloid angiopathy
 - Deposition of amyloid in blood vessel walls → vessels more prone to rupture
 - **Lobar localization**: parietal, occipital lobes
 - **Blood vessels**: leptomeningeal, cerebral cortical arterioles
- Arteriovenous malformations
 - Usually affect children
- Aneurysm, vasculitis, vascular tumours (e.g. hemangioma)

Other causes

- Secondary to ischemic stroke
 - Blood flow blockage → reperfusion → ↑ chance of blood vessel rupture → bleeding into dead tissue (hemorrhagic conversion)

- Posttraumatic
- Coagulopathies
- Sickle cell disease

RISK FACTORS

- Individuals who are biologically male of Asian descent
- Black individuals who are biologically male of African descent
- Heavy **alcohol use**; amphetamines, cocaine abuse, antithrombotic medications; ↓ LDL, cholesterol, triglycerides; previous cerebrovascular insult

COMPLICATIONS

- Hemorrhage enlargement
 - In hemorrhage border
 - Poor prognosis
- Intraventricular, subarachnoid expansion
- Hydrocephalus

SIGNS & SYMPTOMS

- Begin slowly, worsen gradually
- Enlargement of hematoma within few hours, ↑ intracranial pressure
 - Altered consciousness, headache, nausea, vomiting, unequal pupil size
- Fever

Area of brain affected

- **Anterior/middle cerebral artery**: numbness, sudden **muscle weakness**
- **Posterior cerebral artery**: **impaired vision**
- **Broca's area**: **slurred speech**
- **Wernicke's area**: difficulty understanding speech

Focal neurological signs

- **Basal ganglia manifestation**: loss of contralateral sensory, motor functions; homonymous hemianopsia
- **Thalamus**: contralateral loss of

sensory, motor functions; homonymous hemianopsia; aphasia if dominant/neglect if nondominant; narrowed pupils unreactive to light

- **Lobar manifestation:** homonymous hemianopsia; if frontal region, contralateral leg plegia/paresis; seizures
- **Pons:** coma within few minutes of hemorrhage; quadriplegia, miosis/deafness; speaking difficulties when awake
- **Cerebellum:** ataxia; same side face weakness; loss of face, body sensory function; occipital headache, neck stiffness

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Hyperdense blood mass acutely; isodense, ring enhancement appearance in subsequent weeks; hypodense chronically
- Trauma
 - Multifocal bleedings
- Coagulum retracts, edema develops
 - Confused with hemorrhagic infarction

CT angiography

- **Spot sign:** unifocal/multifocal enhancement of contrast; ↑ risk of hematoma expansion

MRI (T2-WI)

- Hyperacute (first 24 hours)
 - Hyperintense center of mass
 - Hypointense periphery, border
- Subacute
 - **Hypointense in > three days:** intracellular methemoglobin
 - **Hyperintense in > seven days:** lysis of red blood cells; extracellular methemoglobin
 - **Chronic:** hypointense; after two weeks

MR angiography

- Vasculitis, arteriovenous malformations, other blood vessel pathology

LAB RESULTS

- Prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count
 - If cause for bleeding diathesis unclear

TREATMENT

MEDICATIONS

- Vitamin K, unactivated prothrombin
 - With anticoagulant usage
- Protamine sulfate
 - For heparin users
- Antipyretics
 - Fever reduction
- Osmotic diuretics (e.g. mannitol)
 - Regulation of ↑ ICP
- Saline
 - Fluid replacement
- Nicardipine/nitroprusside/enalapril/nitroglycerin
 - Hypertension
- Phenytoin/levetiracetam
 - Seizures

SURGERY

- Ventriculostomy
 - Regulation of ↑ intracranial pressure
- If hemorrhage > 3cm/1.2in/lobar of young persons/brainstem compression
 - Craniotomy with clot removal
 - Stereotactic aspiration
 - Endoscopic evacuation

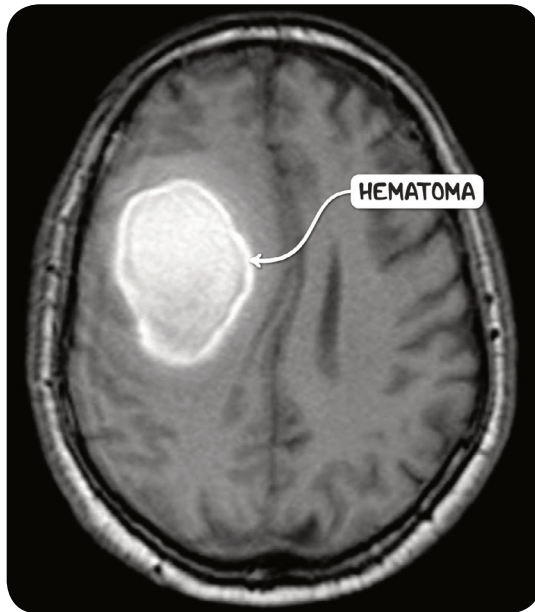


Figure 64.2 A CT scan of the head in the axial plane demonstrating a right-sided, periventricular, intracerebral hemorrhage.

ISCHEMIC STROKE

osms.it/ischemic-stroke

PATHOLOGY & CAUSES

- Decreased blood supply in specific brain area due to blood vessel obstruction → hypoperfusion, tissue hypoxia, infarction
- ↓ blood flow → lack of oxygen, glucose in brain → ↓ adenosine triphosphate (ATP) production, electrochemical gradient → cell death

Two mechanisms of cell death

- **Sodium buildup:** water follows sodium → cell swelling, death
- **Calcium buildup:** creates oxygen radicals → damages mitochondrial, lysosomal lipid membrane → seeping of degradative enzymes, apoptosis-inducing factors → cell death

Two zones

- Ischemic core
 - Brain tissue dies from ischemia within

few minutes of stroke

- Blood flow < 10ml/100g tissue/minute

▪ Ischemic penumbra

- Periphery of affected region preserved due to collateral circulation; chance of survival if blood restored quickly
- Blood flow < 25ml/100g tissue/minute
- Infarction zone spreads if blood supply not restored quickly

TYPES

Five subtypes (TOAST classification)

- Large artery atherosclerosis
- Small artery strokes
- Cardioembolic infarction
 - Formation of emboli in heart → lodging in brain arteries
- Other determined pathology
- Undetermined pathology

CAUSES

Thrombosis

- May lead to obstruction inside blood vessel
- Narrowing of blood vessel **due to atherosclerotic plaque** → gradual ↓ blood flow
- Damage to atherosclerotic fibrous cap → platelet, clotting cascade activation → thrombus formation with sudden stop of blood flow

Embolism

- Four classes based on emboli origin
 - **Cardiac emboli:** atrial fibrillation, rheumatic valve disease, infective endocarditis, dilated cardiomyopathies, left atrial myxoma
 - **Possible cardiac/aortic emboli:** calcification of mitral valve annulus, patent foramen ovale, atheroma in ascending/arch of aorta, atrial septal aneurysm
 - **Arterial emboli:** detachment of blood clot (e.g. atherosclerotic plaque in bigger upstream artery) → emboli travels through blood → lodges in smaller downstream artery
 - **Cryptogenic:** unknown origin of emboli

Lacunar infarct

- Affects small blood vessels of distal vertebral, basilar artery, middle cerebral artery, circle of Willis
 - **Lipohyalinosis:** buildup of hyaline in arterioles wall → hypertrophy of tunica media → progressive narrowing of arterioles until blood flow stops
 - **Microatheromatoma:** narrowing of blood vessel due to debris accumulation within wall

Hypoperfusion

- Lack of blood reaching brain due heart failure, ↓ **cardiac output**
- Most vulnerable
 - Spaces between supply of two arteries (**watershed regions**)

Inflammation of blood vessel wall

- E.g. Takayasu/giant cell arteritis

Moyamoya disease

- Progressive stenosis of cerebral arteries → ischemia

Dissection of artery wall

RISK FACTORS

- Age (esp. > 55)
- More common in individuals who are biologically male
- More common in black individuals of African descent
- Migraine headaches with aura
- Genetics; specific gene loci associated with stroke subtypes
 - **ABO loci:** all subtypes
 - **HDAC9:** large vessel stroke
 - **PITX2, ZFHX3:** cardioembolic stroke
- Hematologic disorders
 - Multiple myeloma, sickle cell disease, polycythemia vera; esp. in younger individuals
- Hypertension, diabetes mellitus, heart diseases, dyslipidemia, hyperhomocysteinemia, smoking, physical inactivity, cocaine abuse

COMPLICATIONS

- Blood reaches infarcted regions through collateral blood vessel/dissolution of occlusive embolus/thrombus; first week

Hemorrhagic transformation

- Ischemia → impaired cellular, metabolic functions of affected region; ↑ permeability of damaged blood vessels → resolved cause of ischemia → restored blood flow → blood extravasation
- Gray matter more commonly affected; large number of collateral vessels worsen reperfusion injury
- Massive cerebral infarction; hyperglycemia; ↓ cholesterol, LDL, IV recombinant tissue plasminogen activator (rtPA): higher risk of hemorrhagic transformation

Cerebral edema

- ↑ intracranial pressure with possible herniation
 - **Cytotoxic:** defective ATP pump, swelling of brain cellular elements due to water

accumulation

- **Vasogenic:** ↑ permeability of blood-brain barrier → ↑ extracellular fluid volume due to ↑ passing of proteins, other macromolecules

Liquefactive necrosis

- **First 48 hours:** edema, paleness of affected region
- **2–10 days:** affected area gelatinous; noticeable border between healthy, damaged tissue
- **3–21 days:** liquefaction of tissue; fluid-filled cavity

Seizures

- Brain injury → ↑ irritability of nervous tissue with neuronal discharges

Deep vein thrombosis

- Esp. immobilized individuals

Pneumonia

- Swallowing mechanism impairment → aspiration pneumonia
- Intubation/ventilatory support → ↑ risk for pneumonia

Dysphagia

- Due to damage of cortex, subcortical structures responsible for swallowing

Dementia

- Altered memory, cognition, behavior due to brain damage

SIGNS & SYMPTOMS

Thrombosis

- Neurological defects

Embolism

- Sudden start of symptoms; maximum defects

Lacunar stroke

- Contralateral, mostly motor/sensory defects; four syndromes
 - **Pure motor stroke:** internal capsule lesion
 - **Pure sensory stroke:** thalamus lesion
 - **Ataxic hemiparesis**
 - **Dysarthria:** speech, swallowing

difficulties; facial weakness; hand weakness, clumsiness (clumsy hand syndrome)

Anterior cerebral artery

- Contralateral hemiparesis (esp. leg, face), sensory deficit; inability to understand, produce speech (left hemisphere); impaired judgment; incontinency

Middle cerebral artery

- Contralateral paresis (esp. face, arm), sensory deficit; inability to understand, produce speech (left hemisphere); hemispatial neglect (right hemisphere); homonymous hemianopsia; deviation of eye to damaged side

Posterior cerebellar artery

- Homonymous hemianopsia
- Cortical blindness (bilateral lesion)
- Midbrain
 - Oculomotor, trochlear palsy → dilated pupil
- Thalamus
 - Sensory loss, impaired memory, altered consciousness
- Posterior cerebellar artery syndrome (PICA)
 - AKA “Wallenberg” syndrome
 - Dizziness, nystagmus; speech, swallowing difficulties
 - **Ipsilateral:** facial sensory loss, Horner’s sign, ataxia
 - **Contralateral:** loss of pain, temperature sensation in limbs

Basilar, vertebral arteries

- Dizziness; gait, vision disorders; dysarthria, dysphagia
- Locked-in syndrome
 - Thrombosis/embolism of basilar artery
 - Plegia of head, body muscles, except eye; only blinking, vertical eye movement possible

DIAGNOSIS

DIAGNOSTIC IMAGING

- If applicable

Noncontrast CT scan

- First few hours

- Affected tissue appears normal
- Later
 - Loss of differentiation between white, grey matter
 - Hypodense parenchyma with sulcal effacement
 - Loss of insular ribbon sign

CT perfusion

- Detection of core, ischemic penumbra

CT angiography

- Find thrombus, embolus in blood vessel/ intra-arterial thrombolysis

MRI

- T1, T2 weighted imaging (see table)
- Diffusion-weighted imaging
 - Shows ischemic stroke early; differentiation from acute, chronic
- Fluid-attenuated inversion recovery (FLAIR) sequence
 - Hyperintense signal within 12 hours

Transcranial Doppler ultrasound

- Visualization of occlusion in middle cerebral artery/intracranial carotid/vertebrobasilar artery

Conventional angiography

- Visualize occlusion; for confirmation of CTA, MRA findings

LAB RESULTS

- Blood tests
 - ↑ cardiac markers in heart disease
 - ↑ erythrocytes in polycythemia vera
 - Toxicology screening (individual suspected of sympathomimetics abuse)
 - ↑ blood glucose level

OTHER DIAGNOSTICS

- Symptoms, neurological changes scoring
- Based on National Institute of Health stroke scale (NIHSS)

ECG

- Detection of myocardial ischemia/atrial fibrillation

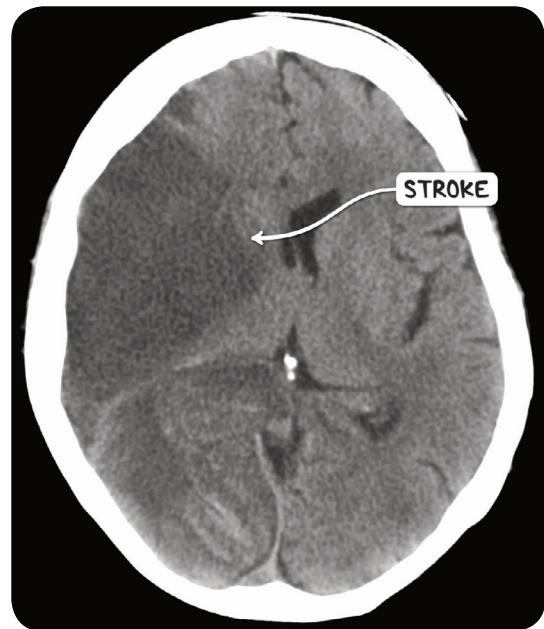


Figure 64.3 A CT scan of the head in the axial plane demonstrating a large ischemic stroke in territory of the middle cerebral artery. The scan was performed three days after the onset of symptoms.

TREATMENT

MEDICATIONS

- Establishment of blood flow in ischemic penumbra
 - **Thrombolytic enzymes:** rtPA; alteplase given within 4.5 hours; after hemorrhagic stroke ruled out
 - **Antiplatelet therapy:** aspirin (325mg orally within 48 hours); other drugs (e.g. clopidogrel/aggrenox)
- Hypertension treatment
 - **IV labetalol/nicardipine:** only if systolic pressure > 220, diastolic > 120 mmHg; except in individuals with vital indications for lowering blood pressure (acute myocardial infarction, kidney failure, dissection of aorta)
- Cerebral edema management
 - **Antipyretic:** if temperature $\geq 40^{\circ}\text{C}/100.4^{\circ}\text{F}$
 - **IV insulin:** hyperglycemia; keep glucose between 140–180 mg/dl (7.8–10 mmol/L)

- Prevention
 - Anticoagulant medications: emboli prevention (e.g. warfarin, aspirin)

SURGERY

- Establishment of blood flow in ischemic penumbra
 - Mechanical embolus removal in cerebral ischemia (MERC) retriever
 - Penumbra system (aspiration, extraction)
 - Solitaire revascularization device, Trevo (stent-retriever systems)
- Cerebral edema management
 - Craniectomy

PSYCHOTHERAPY

- If applicable
- Type of psychotherapy (e.g. group therapy, exposure therapy) with goal of psychotherapy

OTHER INTERVENTIONS

- Cerebral edema management
 - Hyperbaric oxygen therapy: ↑ pure oxygen supply in damaged regions
 - Fluid management: isotonic saline without dextrose
- Protection of airways, prevention of aspiration
 - Head elevation by 30%, nothing by mouth/*nil per os* (NPO) status
- Prevention
 - Control risk factors (for atherosclerosis): e.g. smoking, hypertension, diabetes, aspirin use; carotid endarterectomy
 - Lifestyle alteration: exercising, appropriate diet

MRI FOR ISCHEMIC STROKE

	T1 WEIGHTED IMAGING (T1-WI)	T2 WEIGHTED IMAGING (T2-WI)	NOTES
HYPERACUTE	Hypointense signal (16-24 hours)	Absent flow void signal (2 hours); Hyperintense signal from affected region (8 hours)	
ACUTE (1-7 DAYS)	Hypointense	Hyperintense	T1, T2-WI masked by reperfusion in 24-48 hours → fogging phenomenon
SUBACUTE (7-21 DAYS)	Hypointense	Hyperintense	
CHRONIC (> 21 DAYS)	Hypointense	Hyperintense	

SACCULAR ANEURYSM

osms.it/saccular-aneurysm

PATHOLOGY & CAUSES

- Asymmetrical ballooning of blood vessel wall
- Bifurcation of arteries common place esp. on circle of Willis due to weakness of wall, turbulent blood flow
 - Anterior communicating (most common); posterior communicating; middle cerebral; internal carotid; basilar artery tip

TYPES

Type A

- Blood vessel wall with endothelium, linear smooth muscle

Type B

- Disorganization of smooth muscle, thickening of wall

Type C

- Hypocellular wall with thickening of intima/ luminal thrombosis

Type D

- Hypocellular wall coated with thin thrombosis layer

CAUSES

- Inborn defect of arteries, lack of external lamina, tunica media → hemodynamic stress over years → gradual ballooning of blood vessel wall, thickening of intima, adventitia

RISK FACTORS

- More common in individuals who are biologically female, > 50 years (due to estrogen deficiency)
- Diseases associated with aneurysm
 - Ehler-Danlos syndrome, pseudoxanthoma elasticum, lupus, autosomal dominant polycystic kidney

disease (ADPKD), bacterial endocarditis, fibromuscular dysplasia

- Familial predisposition; smoking; alcohol, cocaine use; hypertension; trauma

COMPLICATIONS

- Warning leaks
 - May precede aneurysm rupture; strong headaches, photophobia, nausea/ vomiting
- Rupture → subarachnoid hemorrhage
 - Apex of aneurysm/atheromatous plaque edge
 - ↑ risk in smokers, individuals with migraines, elderly, affection of posterior circulation, larger size
- Ischemia
 - Thrombus forms within aneurysm → detachment of small particles (emboli) → emboli lodges → ischemia of downstream tissue
- Multiple aneurysms

SIGNS & SYMPTOMS

- May be asymptomatic if small
- Mass effect symptoms due to size
 - Anterior communicating artery: both leg weakness with positive Babinski sign
 - Posterior communicating, internal carotid artery: headaches with palsy of oculomotor nerve
 - Left middle cerebral artery: inability to understand, produce speech
 - Right middle cerebral artery: contralateral field vision loss

DIAGNOSIS

DIAGNOSTIC IMAGING

CT/MR angiography

- Detect aneurysms > 2mm

TREATMENT

SURGERY

- Endovascular management
 - Aneurysmal coiling with thrombosis → endothelialization across aneurysm neck → prevents rebleeding, regrowth
 - In development: stent-assisted, balloon-assisted coiling; disruptors, flow diverters
- Surgical clipping

OTHER INTERVENTIONS

- Regular monitoring with CTA/MRA
- Avoid smoking, alcohol, drugs, excessive strain

SUBARACHNOID HEMORRHAGE (SAH)

osms.it/subarachnoid-hemorrhage

PATHOLOGY & CAUSES

- **Bleeding** into space between pia mater, arachnoid membrane
- Injury/spontaneous event → rupture of blood vessel in subarachnoid space → release of blood into cerebrospinal fluid (CSF) → rapid ↑ intracranial pressure

- Smoking; hypertension; alcohol, cocaine abuse
- Diseases associated with saccular aneurysm (e.g. blood vessel disorders, Ehlers–Danlos syndrome, Marfan syndrome, polycystic kidney disease)
- Sickle cell disease
- Coagulopathies

CAUSES

- **Traumatic:** head injury (e.g. bridging vein tear)
- **Spontaneous:** arterial origin (more common)
 - Rupture of saccular “berry” aneurysms (e.g. anterior half of circle of Willis)
 - Arteriovenous blood vessel malformations

RISK FACTORS

- More common in individuals who are biologically female, elderly

COMPLICATIONS

- **Vasospasm**
 - Delayed ischemia; 4–11 days after SAH
 - Blood clot lysis → release of spasmogenic substances (e.g. endothelin), ↓ production of nitric oxide → vasospasm due to smooth muscle contraction → brain ischemia
- **Hydrocephalus**
 - Clogging of CSF drainage
- **Rebleeding**
 - May occur two weeks after SAH
 - ↑ tendency in individuals with

hypertension, anxiety, seizures post-SAH

- Associated with ↑ mortality, neurological damage

- Sympathetic hyperactivity due to ↑ intracranial pressure, SAH (“sympathetic surge”) → sudden, life-threatening ↑ of blood pressure due to vasoconstriction
- ↑ plasma adrenaline levels due to sympathetic hyperactivity → arrhythmias
- Over-action of sympathetic nervous system → pulmonary vasoconstriction → ↑ capillary permeability, pressure → neurogenic pulmonary edema
- Hyponatremia
- Meningitis (irritation from presence of blood)
- Seizures

SIGNS & SYMPTOMS

- Area of brain
 - **Anterior/middle cerebral artery:** numbness, sudden muscle weakness
 - **Broca’s area:** slurred speech
 - **Wernicke’s area:** difficulty understanding speech
- ↑ intracranial pressure
 - **Thunderclap headache:** “**worst ever**” headache; may be only symptom
 - Nausea, vomiting
- Altered consciousness; coma, confusion, seizures
- **Meningeal irritation:** bleeding into subarachnoid space filled with CSF → blood degradation → irritation of meninges, development of aseptic meningitis
 - Neck pain, stiffness
 - **Positive meningeal signs:** Kernig’s (pain generated by knee extension from 90°); Brudzinski’s (forced neck flexion → spontaneous knee, hip flexion)
 - Photophobia
- Focal neurological signs
 - Posterior communicating artery aneurysm rupture/brain herniation due to ↑ intracranial pressure → oculomotor nerve paralysis → ipsilateral ptosis; eye pointed down, out; mydriasis, loss of pupillary light reflex

- ↑ intracranial pressure → abducens nerve paralysis → eye pointing out → diplopia

DIAGNOSIS

DIAGNOSTIC IMAGING

Noncontrast CT scan

- Fisher scale grading
 - **Group 1:** no hemorrhage
 - **Group 2:** blood depositions < 1mm, without blood clots
 - **Group 3:** blood depositions > 1mm, with localized clots
 - **Group 4:** diffuse/lack of subarachnoid hemorrhage with extension to ventricles, brain parenchyma
- Hydrocephalus
 - “Mickey Mouse” ventricular system appearance

MRI

- Visualize arteriovenous malformations (not detected by angiography)

Digital-subtraction cerebral/CT/MR angiography

- Visualize aneurysm

LAB RESULTS

- Identify hematologic abnormalities
- **PT, aPTT:** identify coagulopathies
- ↑ troponin, if heart abnormalities present

OTHER DIAGNOSTICS

- **Lumbar puncture**
 - ↑ erythrocytes in all three samples
 - CSF centrifugation: **yellow coloration** due to erythrocytes breakage, release of heme (“xanthochromia”); positive 3–4 weeks after SAH
- Physical examination
 - Characteristic neurological presentation; fever; tachycardia; fundoscopy (optic disc swelling, retinal hemorrhages)

ECG

- ↑ QRS, QT intervals; ↓ PR intervals; U waves; dysrhythmias

TREATMENT

MEDICATIONS

- **Antihypertensive therapy:** beta-blockers; hydralazine, calcium channel blockers; ACE inhibitors
- **Intracranial pressure treatment:** osmotic, loop diuretics
- **Prior all procedures:** IV midazolam (initial treatment)
- **Vasoconstriction treatment:** calcium channel blocker (e.g. **nimodipine**), recombinant tissue plasminogen activator
- **Seizure treatment:** phenytoin/phenobarbital
- **Pulmonary edema treatment:** diuretics, dobutamine

SURGERY

- **Aneurysm treatment:** endovascular coiling (with aneurysm obliteration), craniotomy (with aneurysm neck clipping, coiling)
- **Vasoconstriction:** aspiration/irrigation of blood clot during clipping process, CSF drainage, transluminal balloon angioplasty
- **Hydrocephalus:** temporary/serial lumbar puncture for CSF drainage, permanent ventricular shunt, ventriculostomy

OTHER INTERVENTIONS

- Vital sign stabilization
- Intubation if comatose, heart monitoring (initial treatment)
- Keep blood pressure < 140mmHg to avoid rebleeding

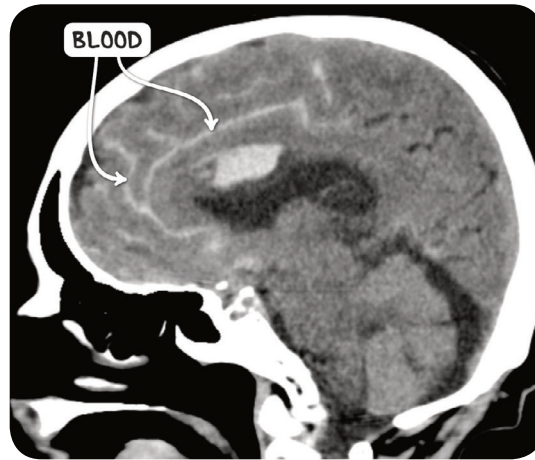


Figure 64.4 A CT scan of the head in the sagittal plane demonstrating high signal in the sulci of the frontal lobe, consistent with a subarachnoid hemorrhage.

SUBDURAL HEMATOMA (SDH)

osms.it/subdural-hematoma

PATHOLOGY & CAUSES

- Intracranial bleeding with blood accumulation between dura mater, arachnoid membrane
- Head trauma → **tearing of venous blood vessels**/small cortical arteries → blood accumulation → limited blood mass expansion due to adherent dural attachments → brain tissue compression

TYPES

Acute

- Slow blood outflow into subdural space due to low pressure in bridging veins

Subacute

- Combination of fluid, clotted blood

Chronic

- Caused by **minor trauma**/inflammation
- More common in **elderly**
- Head trauma with small bleeding, dural border cell damage → inflammation, unsuccessful attempt to repair border cells with formation of granulation tissue → encapsulation; development of blood vessels within new membrane → erythrocytes, plasma exudation from leaky capillaries to encapsulated space → recurrent bleeding with expansion

CAUSES

- **Head trauma** (most common)
- Acceleration-deceleration (coup-contrecoup injury)
- Acceleration of body → sudden stop with forwarding momentum carrying brain → impacts front of skull → backward brain movement → impacts back of skull → bridging veins tear
- Intracranial hypotension
 - ↓ CSF due to lumbar puncture/lumboperitoneal shunt → ↑ traction

of brain on surrounding structures → bridging veins tear

- Shaken baby syndrome
- Spontaneous
 - Vascular malformations
- Neurosurgical procedure complication

RISK FACTORS

- **Brain atrophy** elderly
 - ↑ bridging veins stretch
- Infants, alcohol abusers
 - Thinner wall of bridging veins
- Epilepsy, anticoagulant drugs, thrombocytopenia

COMPLICATIONS

- Liquefaction of granulation tissue in chronic subdural hematoma (subdural hygroma) → ↑ protein → expansion of mass due to water drawn by osmotic pressure → mass effect brain injuries
- ↑ intracranial pressure → supratentorial, infratentorial herniation of brain
- Progressive dementia in chronic subdural hematoma

SIGNS & SYMPTOMS

- Loss of consciousness after trauma/in ensuing days due to hematoma expansion
- Bleeding characteristics
 - **Hemispheric**: most common
 - **Interhemispheric**: altered consciousness, headache, hemiparesis
- Physical examination
 - Broken basilar skull: periorbital ecchymosis (raccoon eyes), retroauricular ecchymosis (Battle's sign)
 - CSF rhinorrhea/otorrhea
- Acute subdural hematoma
 - Neurological presentation in 48–72 hours

- May be comatose/awake
- Sudden, severe headache with nausea, vomiting; unequal pupils; difficulties in speech, swallowing; palsies of cranial nerves
- Subacute
 - Presents 2–14 days
- Chronic
 - Present 14 days after injury
 - Impaired cognitive skills, altered consciousness, headaches, contralateral/ipsilateral hemiparesis (depends on hematoma location), hemianopsia, optic disc swelling

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- **Acute:** **crescent-shape** hyperdense blood collection
- **Subacute/chronic:** isodense/hypodense crescentic mass

MRI

- **T2-WI (acutely):** hypointense blood clot due to deoxyhemoglobin
- **T1, T2-WI (in following weeks):** bright appearance; deoxy → methemoglobin
- **T1-WI (months later):** hypointense clot due to hemosiderin remains

MR/CT angiography

- Spontaneous SDH

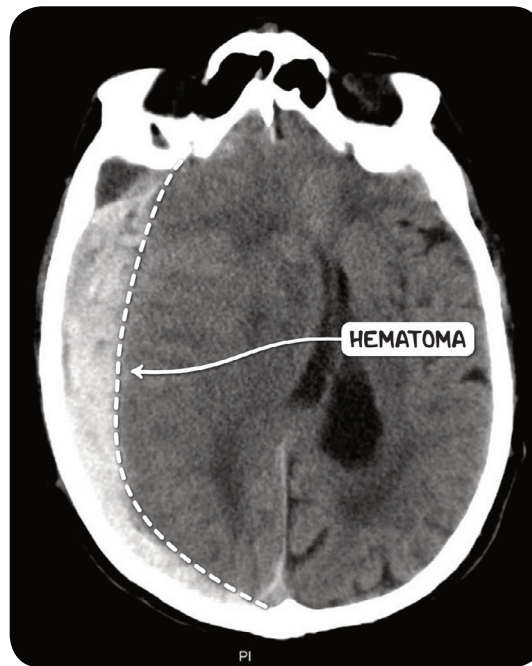


Figure 64.5 A CT scan in the axial plane demonstrating a large, right-sided, subdural hematoma. The hematoma has a classical crescentic shape.

TREATMENT

MEDICATIONS

- Diuretics
 - ↓ intracranial pressure
- Vitamin K/factor VIII inhibitor activity bypassing agent (FEIBA)/frozen plasma
 - Anticoagulation reverse; ↓ risk of hematoma enlargement; individuals undergoing surgery

SURGERY

- If clot thickness > 10mm, midline shift > 5mm, intracranial pressure > 20mmHg
 - Burr hole, craniotomy, decompressive craniectomy, blood vessel ligation

OTHER INTERVENTIONS

- Nonsurgical treatment based on Glasgow coma score (GCS); clot thickness (< 10mm); neurological examination; stable/deteriorated condition; comorbidities, associated injuries; age

TRANSIENT ISCHEMIC ATTACK (TIA)

osms.it/transient-ischemic-attack

PATHOLOGY & CAUSES

- Short-lasting neurological dysfunction due to transient focal ischemia, without infarction
- Blood vessel occlusion/stenosis → ↓ blood flow in affected region → neurological dysfunction

CAUSES

- **Adults:** thrombosis, hypoperfusion, emboli
- **Children:** congenital heart defects with thrombosis, coagulopathies, idiopathic progressive arteriopathy of childhood (Moyamoya disease)

RISK FACTORS

- More common in black individuals of African descent who are biologically male; ↑ risk with age
- Family history, hypertension, diabetes, obesity, obstructive sleep apnea, ↑ low-density lipoprotein (LDL), ↓ high-density lipoprotein (HDL), atherosclerosis, cocaine abuse, smoking

COMPLICATIONS

- Recurrent TIA
- Ischemic stroke

SIGNS & SYMPTOMS

- **Duration:** few minutes to one hour
- ↓ **flow in large arteries (few minutes)**
- Numbness/paresis
 - Face, cheek, tongue, arm, hand, leg
- Aphasia
 - If dominant hemisphere affected
- Hemispatial neglect

- If nondominant hemisphere affected
- Distal vertebral artery
 - Dizziness, difficulty speaking, double vision
- Mid-basilar artery
 - Dizziness, paresis affecting both legs/arms

Embolic TIA (> one hour)

- Anterior cerebral circulation
 - Symptoms depend on blood vessel lodged
 - **Middle cerebral artery:** contralateral hemiplegia; aphasia if dominant hemisphere; hemispatial neglect if nondominant
 - **Branches of middle cerebral artery:** numbness/motor function loss; face, arm, leg
 - **Ophthalmic artery:** amaurosis fugax; transient monocular/binocular vision loss
- Posterior cerebral circulation
 - Dizziness, focal hearing loss, speech difficulties, double vision, hemi/quadrantanopia, face/body numbness
 - **Basilar artery:** thalamus, subthalamus, medial midbrain, reticular activating system → stupor/coma

DIAGNOSIS

DIAGNOSTIC IMAGING

CT/MR/conventional catheter angiography

- Occlusion within blood vessel

Diffusion-weighted MRI

- Ischemic regions corresponding to neurologic symptomatology
- Changes seen within first few hours of symptoms

Perfusion-weighted MRI

- ↓ tissue blood flow

Neck Doppler ultrasound

- Evaluate carotid stenosis

LAB RESULTS

- Hypoglycemia, hyponatremia, thrombocytosis: rule out conditions that mimic TIA

OTHER DIAGNOSTICS

- See mnemonic
 - **ABCD2 score**: evaluate risk for possible ischemic stroke (can occur two days after TIA)

**MNEMONIC: ABCD2****Evaluating ischemic stroke risk**

Age
Blood pressure
Clinical features
Duration of symptoms
Diabetes

TREATMENT**MEDICATIONS**

- Antiplatelet (noncardioembolic TIA)
 - Aspirin/extended-release dipyridamole/ aspirin + clopidogrel
- Anticoagulation
 - **Atrial fibrillation**: low-molecular-weight heparin
 - **Heart thrombus**: in acute myocardial infarction/rheumatic mitral valve; warfarin + direct acting oral anticoagulants (e.g. apixaban)
- Diuretics, angiotensin-converting enzyme (ACE) inhibitors
 - Blood pressure control
- Statins
 - Cholesterol management

SURGERY

- Same side carotid stenosis/TIA
 - Carotid endarterectomy

OTHER INTERVENTIONS

- Mediterranean diet